Psychiatric Case Series

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Dear Doctor,

Greetings from Micro Labs Limited.

We are happy to bring you the "PSYCHIATRY Case series", which contains the latest and most interesting cases in the field of Psychiatry.

This issue contains

Case report on Auditory Charles Bonnet syndrome and psychosis

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Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback

Best Regards,

Medical Team,

1. Case report on Acute Mania in Fanconi–Bickel Syndrome

Introduction:

This case suggests a relationship between mood and metabolism, with glucose processing playing a role in mood manifestations.

Fanconi–Bickel syndrome is a carbohydrate metabolism disorder caused due to glucose transporter 2 (GLUT2) transporter defect (Figure 1). Fanconi and Bickel originally reported Fanconi–Bickel syndrome in 1949. The major characteristic of FBS was accumulation of glycogen in the kidney and liver. Variants in the SLC2A2 gene have been discovered in FBS instances, including missense, nonsense, frame-shift (fs), in-frame indels, splice site, and compound heterozygous.[1] Psychiatric diseases and metabolic abnormalities have long been thought to be related. This was supported by the fact that individuals with psychiatric disorders were more likely to have metabolic syndromes linked to blood glucose imbalance. However, it's still uncertain if metabolic pathways and mood were directly related. Patients with type 1 diabetes also have a higher risk of mood and psychotic problems. A clear pathophysiological explanation relating mood or cognitive impairments especially to GLUT2 function was not clear. This was supported by the absence of behavioral research that particularly look at how genetic and metabolic alterations affect behavior.[2] The patient with FBS described in this case study had two separate admissions to a psychiatric inpatient, with symptoms similar to acute mania commonly observed in bipolar I disorder.

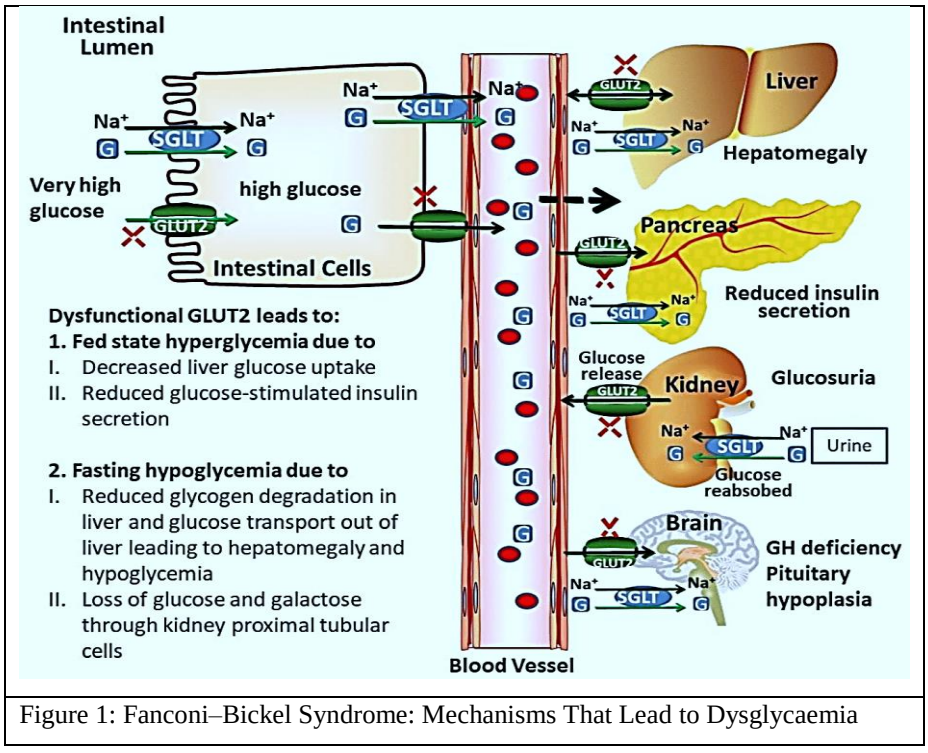

Figure 1: Fanconi–Bickel Syndrome: Mechanisms That Lead to Dysglycaemia

Case Presentation:

A 28-year-old man with consanguineous parents, diagnosed with FBS as a kid, presented to the psychiatric emergency department with severe acute mania. He has chronic renal disease,

heart problems, hepatomegaly, postprandial hyperglycemia, and fasting hypoglycemia that necessitate frequent meals. He exhibited impulsivity and aggression in early adolescence. The individual's first psychiatric hospitalization happened at the age of 21 due to a severe major depression. Despite a history of intermittent cocaine, benzodiazepine, and cannabis use, the patient was not taking drugs during his hospitalizations. He was hospitalized to the psychiatric unit twice. The patient's first admission was preceded by a violent incident in which he repeatedly punched his father and threatened to kill him. In the psychiatric emergency room, he regularly assaulted other patients and staff. The patient received emergency treatment including haloperidol, lorazepam, fluphenazine, chlorpromazine, and diphenhydramine. Upon admission, the mental status assessment revealed extreme irritability, grandiosity, recurring aggressive conduct, psychomotor agitation, racing thoughts, and paranoia. Treatment began with fluphenazine 10 mg twice a day and valproic acid 500 mg in the morning and 1,000 mg at night. Fluphenazine decanoate was also given. After 10 days, the patient recovered to euthymia and was discharged. Six days following discharge, the patient returned to the psychiatric emergency room. His father stated that he did not continue taking fluphenazine and valproic acid after being discharged. He stopped sleeping and returned to a manic state marked by irritability, grandiosity, impulsivity, psychomotor agitation, aggressive conduct, and paranoia. In a psychiatric emergency department, a patient seemed calm, asked a nurse a question, and then violently punched the nurse and other personnel without reason. During his second inpatient admission, he was prescribed fluphenazine 10 mg twice a day and valproic acid 500 mg in the morning and 1,000 mg at night. After 12 days, the patient recovered to euthymia and was discharged.

Discussion:

Glycogen Storage Disease XI, or FBS, was first identified by Fanconi and Bickel. It was a rare illness of glucose metabolism caused by several known loss-of-function mutations in SLC2A2, the gene that codes for the glucose transporter 2 (GLUT2). This study demonstrated that GLUT2 loss in FBS may result in an overactive PVT network, causing abnormal excitatory input to the amygdala and nucleus accumbens, among other structures (Figure 2). In the present case, treatment of acute mania began with fluphenazine 10 mg twice day and valproic acid 500 mg in the morning and 1,000 mg at night (valproic acid blood level of 99). Fluphenazine decanoate was also given.[2] In the previous study on first manic attack, the patient was treated with zuclopenthixol acetate, 5 mg biperiden and 10 mg haloperidol was given by

intramuscular route. Zuclopenthixol decanoate every 2 weeks and 2 × 10 mg olanzapine prescribed.[3]

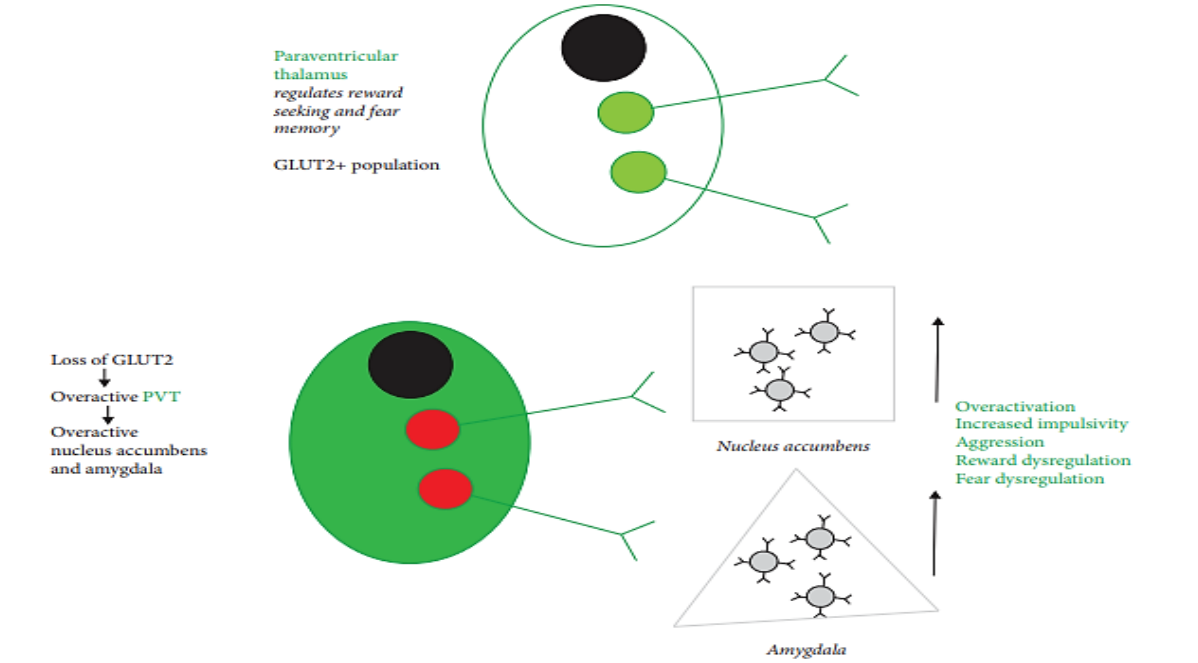

Fig 2. GLUT2 loss in FBS may result in an overactive PVT network, causing abnormal excitatory input to the amygdala and nucleus accumbens, among other structures.

Conclusion:

This was the first report of a possible neuropsychiatric manifestation of FBS. The hypothesis of how mania can be explained by FBS suggested that reduced GLUT2 function might cause overactivity of the PVT, leading to increased excitatory input to brain areas such as the amygdala and nucleus accumbens, which were known to be abnormally active during mania. This case demonstrated a relationship between mood and metabolism, with glucose processing playing a role in mood manifestations.

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2. Case report on idiopathic calcification of the basal ganglia associated with symptoms of obsessive-compulsive disorder

When a patient has calcified brain lesions, Idiopathic basal ganglia calcification (IBGC) should be considered as part of the differential diagnosis.

Introduction:

Idiopathic basal ganglia calcification (IBGC), also known as Farh's disease, is a rare neurodegenerative disease that causes calcification in several brain locations. IBGC is a progressive illness, with aberrant calcium deposition starting in the third decade of life and neurological symptoms appearing two decades later. Movement disorders were the most prevalent IBGC symptoms, accounting for 55% of all symptomatic patients. About 57% of the patients had parkinsonism, 19% had chorea, 8% had tremors, 8% had dystonia, 5% had athetosis, and 3% had facial dyskinesia. About 40% of patients have psychiatric features as their predominant symptoms. Mood disorders, cognitive decline, and psychotic symptoms were frequent psychiatric features.[1] Importantly, Obsessive-compulsive disorder (OCD) was one of the most common clinical characteristics of neurological illnesses and plays a significant role in their pathogenesis.[2] However, the pathophysiology of IBGC's psychiatric manifestations was unknown, and symptoms vary in severity between cases.[1] This case report described a case of IBGC who experienced obsessive thoughts triggered by trivial events, with repeated suicide attempts.

Case Presentation:

A 55-year-old man presented with high anxiety over his kidney cancer recurrence. At the age of 47-years, the patient underwent surgery for left kidney carcinoma and had an easy recovery. During health check-up, it was found haematuria which further increased his fear about recurrence. Despite a thorough evaluation revealed no recurrence, the patient continued to experience anxiety. Three months following the check-up, the patient was diagnosed with depression at a psychiatric clinic and began pharmacotherapy. The patient's mental instability caused him to fear of losing his job. The next day, the patient attempted suicide by hanging. When the patient arrived to hospital, he resisted medical procedures with his eyes closed, suggesting a dissociative state. Despite a major suicide attempt, the patient reported only vague anxiety and no depression symptoms. The patient was admitted to the psychiatric department for treatment and evaluation for psychiatric symptoms. Head CT revealed bilateral calcifications of the basal

ganglia, thalamus, and dentate nucleus for the first time (Figure 1).

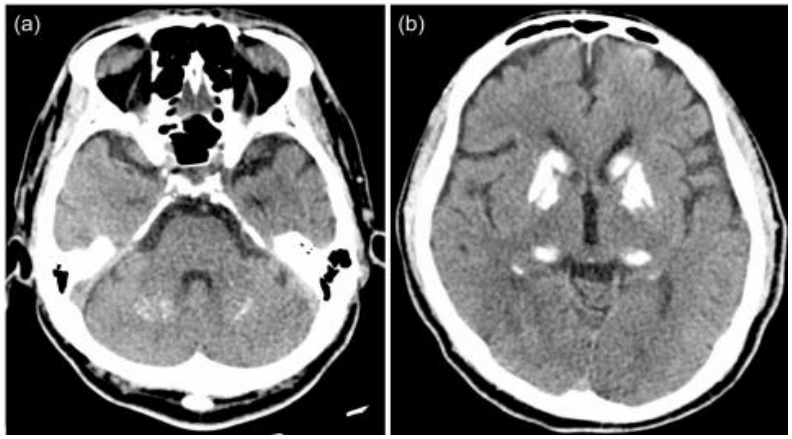

Figure 1: A brain computed tomography image demonstrating bilateral calcification of the dentate nucleus, thalamus, and basal ganglia.

The patient's anxiety and confusion were treated with antidepressants and antipsychotics, which led to improved mental health. He was diagnosed with depression and was discharged from the hospital two months later, allowing him to return to work. The treatment for depression included olanzapine (2.5 mg), sulpiride (100 mg), aripiprazole (3 mg), and valproic acid (200 mg). However, the antipsychotics produced drug-induced parkinsonism, including tremors and brachybasia. The patient also reported physical anxiety and emotional instability. The patient wrote a suicide note for his wife and tried suicide by overdosing on sleeping tablets. The family discovered him unconscious in bed so they took him to hospital. Prior to admission, the patient had been taking aripiprazole (0.75 mg), valproic acid (200 mg), biperiden (2.5 mg), flunitrazepam (2 mg), and brotizolam (0.25 mg). Following this overdose event, all drugs were temporarily stopped, and the patient was followed up. On admission, the neurological examination revealed a minor bilateral tremor in the hands as well as a masked face. A brain CT scan showed symmetrical calcification in the basal ganglia, thalamus, and dentate nucleus, with a little wider area than previous (Figure 2). On the ninth day of admission, he became upset and screamed that he had been deceived into being hospitalized. The patient had a stupor with closed eyelids the next day. The patient apologized for his distress once the symptoms resolved. Magnetic resonance imaging (MRI) and dopamine transporter (DAT) imaging were used to evaluate the patient's psychiatric symptoms and parkinson's disease. The MRI revealed calcification in the same locations as the CT scan, with no further aberrant findings. DAT scans revealed lower bilateral putaminal specific binding ratios. The genetic study confirmed the clinical diagnosis of IBGC, which was caused by a heterozygous

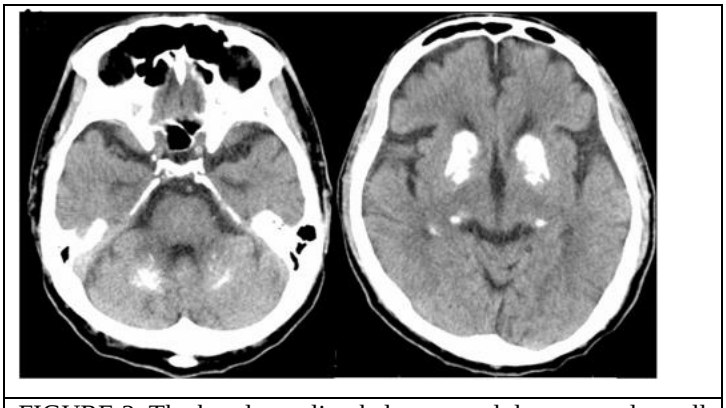

FIGURE 2. The basal ganglia, thalamus, and dentate nucleus all showed symmetrical calcification on the brain computed tomography (CT) scan, with a marginally increased calcification area.

SLC20A2 nonsense mutation. IBGC was shown to be associated with previously identified obsessive-compulsive and anxiety symptoms. The patient's psychiatric symptoms resolved spontaneously, and valproic acid (200 mg) was continued to prevent mood instability. He was discharged from the hospital after a month.

Discussion:

IBGC is characterized by calcification of the bilateral basal ganglia. It often affects middle-aged people with neurological and psychological problems. CT was effective for identifying calcifications. Clinically, this patient showed signs of uncontrollable obsessions brought on by insignificant incidents, which resulted in suicide attempts and motor symptoms such as parkinsonism.[1] Previous examples of IBGC with psychiatric symptoms reported mood problems, cognitive impairment, and psychotic symptoms.[3] Conversely, a number of reports of IBGC patients exhibiting obsessive-compulsive symptoms were reported.[4] The patient in this case had a range of symptoms, including hopelessness, anxiety, dissociative symptoms, and suicidal thoughts, all of which were caused by OCD.[1]

Conclusion:

This case study demonstrated the value of thorough clinical exams and brain imaging in the assessment of patients who presenting with motor and psychiatric complaints. IBGC need to be considered in the differential diagnosis, especially when there were calcified brain lesions.

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3. Case Report on the Use of Electroconvulsive Therapy to Treat Catatonia Associated with Lewy Body Dementia

The present case demonstrates that Electroconvulsive Therapy (ECT) should be considered for refractory catatonia caused by LBD, especially when other treatments are unsuccessful.

Introduction:

Lewy body dementia, which includes dementia with Lewy bodies (DLB) and Parkinson's disease dementia (PDD), is the second most frequent neurodegenerative dementia in older adults, accounting for 15-20% of all dementia cases in pathological studies. Clinically, the prevalence was substantially lower: PDD prevalence was estimated to be 3.6% and DLB prevalence to be 4.2-5% of all dementia patients.[1] The primary basis for the diagnosis was the onset and start of certain symptoms, including the onset of motor symptoms and dementia. In particular, the diagnosis was consistent with LBD if the dementia symptoms start a year before the motor symptoms. Catatonia is a prominent sign of the condition, posing challenges for diagnosis, treatment, and on its own can be potentially lethal. It was a combination of neuropsychiatric symptoms that can occur in both psychiatric and neurological disorders. Catatonia caused by psychiatric diseases can be effectively treated with benzodiazepines and electroconvulsive therapy. Treating catatonia caused by neurological disorders can be challenging, especially when associated with Lewy Body Dementia (LBD). [2] In this case report, a 78-year-old woman with LBD, Bipolar I, and depression had twelve ECT sessions to treat catatonia that did not respond to benzodiazepine therapy.

Case Presentation:

A 78-year-old female patient had a medical history of essential hypertension, intrinsic eczema, mixed hyperlipidaemia, slow transit constipation, bipolar I, and unidentified catatonia. Before being admitted to the psychiatric health facility, the patient first displayed signs of depression and suicide ideation that persisted for several weeks. During the initial assessment, the patient was dysphoric and crying, with recurrent suicide thoughts, physical preoccupations, and anxiety. The patient exhibited tangential thinking, anger, and delusions of being "dead-brained" or "dead person". The patient attempted suicide while in the facility. After a few months, the patient's condition worsened, including a slow shuffling gait, cogwheel stiffness in the upper limbs, and trouble eating. The clinical presentation of dementia symptoms followed by parkinsonism more than a year after the beginning of cognitive impairment was the basis for the diagnosis of Lewy Body Dementia (LBD). Her cognition fluctuated every day, indicating LBD. She suffered Capgras illusions, which were common of neurodegenerative diseases.

Catatonia symptoms, including mutism, low-grunting sounds, purposeless motions, repeated hand rubbing, facial grimacing, motor hesitation, and poor oral intake, appeared early after LBD diagnosis. She needed assistance with her activities of daily living (ADLs). The patient was started lorazepam 4 mg orally as a total daily dosage. The patient's psychotic symptoms grew worse as she developed nihilist illusions of "being dead" and "disappearing," as well as increased paranoia and suspicion of imposters among the unit staff and providers. To address the patient's poor reaction to quetiapine, olanzapine, and clozapine, as well as their intolerance to chlorpromazine, pimavanserin (34 mg orally daily) was prescribed for psychosis with parkinsonism. The patient's psychotic symptoms decreased significantly, although her concentration, focus, and attention varied throughout the day, as expected with LBD. Rivastigmine was introduced to treat cognitive fluctuations and behavioral dysregulation, with limited efficacy. The patient experienced paranoid delusions and agitation after using Sinemet® (carbidopa/levodopa 25/100 mg) twice daily for parkinsonism. Amantadine (100 mg orally daily) was then used to treat both parkinsonism and catatonia. Initially, she reacted positively with less parkinsonism and a lower Bush-Françis Catatonia Scale (BFCS) score. However, when the dose was raised, she developed suicidal ideations (SI). The trial of Sinemet® (25/100 mg twice daily) for rigidity and akinesia was discontinued due to increasing catatonic rigidity, mutism, verbigeration, and finger play. Amantadine 100 orally daily was resumed. Symptoms of catatonia reappeared including nonverbal behavior, increased grasp, lip pursing, finger play, stiffness, and mitgehen. The Bush-Françis score was 1-14 8, 1-23, 13. The patient's intermittent pattern of catatonic-like behaviors intensified, including continual finger play, mutism, extreme negativism, repeated lip pursing, grimacing, vocalization "pee pee poo poo," and fixed downward stare. The Bush-Françis score reached to 27. At this period, alternative remedies were planned and investigated. ECT therapy was started biweekly utilizing a bi-temporal short pulse technique, resulting in sufficient seizures. After three ECT treatments, there was significant improvement in motor activity, reduced bradykinesia, increased orientation, and full resolution of catatonia. To reduce the risk of delirium and cognitive deterioration associated with ECT, sessions were reduced from twice a week to weekly. The BFCS score after six sessions was 5 (Table 1). After 12 sessions, the patient's BFCS score was decreased to zero and ADLs improved, requiring

Category	Score	Associated symptoms
Mutism	1	Verbally unresponsive, incomprehensible whisper.
Staring	1	Poor eye contact, repeatedly gaze less than 20 seconds between shifting of attention and decreased blinking.
Mitgehen	3	As defined.

Table 1: Pertinent Bush-Françis Catatonia Scale scores after six sessions of ECT therapy

less help. The patient's communication skills improved, and her motor movement became less stiff and repetitive. She was able to complete ADLs independently and interact with staff more effectively. Prior to ECT therapy, the patient was taking Sinemet®, amantadine, pimavanserin, and sertraline, but her catatonic symptoms did not improve much. Follow-up strategies include continuing ECT therapy to lower BFCS to zero, then discontinuing it and pursuing diagnostic imaging with a DAT scan.

Discussion:

ECT is often used to treat resistant depression or psychiatric illnesses with life-threatening symptoms including suicidality or severe psychosis. Although there were no clear contraindications, individuals with recent myocardial infarction, severe hypertension, or elevated intracerebral pressure should proceed with care. The patient experienced limited success with previous pharmacotherapies, prompting the choice to use ECT. This case supports the use of ECT in refractory catatonia linked to LBD, especially in cases when pharmacotherapies were unsuccessful.[2] Vaquerizo-Serrano J et al. on catatonia in autism spectrum disorders (ASD) revealed that benzodiazepines, antipsychotics, and electroconvulsive therapy (ECT) have all been used to treat ASD with catatonic symptoms. The systematic review's findings suggested that ECT might assist treat catatonic symptoms.[3]

Conclusion:

This case report with LBD and refractory catatonia highlighted the ineffectiveness of conventional therapies for catatonia linked with dementia. After unsuccessful pharmacotherapies, the patient underwent ECT treatment. Following 12 sessions, there was considerable improvement in catatonic symptoms as measured by the Bush-Françis Catatonia Scale. Improved Parkinsonian symptoms emphasized the treatment's dual benefits for dementia-related illnesses, notably LBD. This example suggested using electroconvulsive therapy (ECT) to treat refractory catatonia caused by LBD, especially when other treatments were ineffective.

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4. Case report on thiamine deficiency neuropathy in a malnutrition patient caused by Melancholic depression

Introduction:

This case highlighted how crucial it was to identify and treat thiamine deficiency in people suffering from severe depression, especially in those with significant weight loss and displaying melancholic symptoms.

Depression is now recognized as a systemic disorder caused by several biological mechanisms, such as inflammation, hypothalamic-pituitary-adrenal (HPA) axis dysfunction, and imbalances in neurotransmitters and neurotrophic systems.[1] In 2015, the World Health Organization (WHO) reported that 4.4% of the world's population suffering from depression. According to the DSM-5 classification, "melancholic features" indicate a severe depressive episode. The main clinical symptoms of melancholic depression are psychomotor retardation and lack of emotional responsiveness. Other symptoms may include psychomotor agitation, early awakening, and loss of appetite/weight. Depressive episodes can cause considerable weight loss or undernutrition.[2] This case described an unexpected neurological complication caused by a melancholy condition and dietary insufficiency.

Case Presentation:

A 36-year-old woman exhibited apathy and food refusal. The patient's family characterized her as extremely emotional and sensitive. The incident began 2 months ago with a quick progression towards isolation, irritability, and religious worries. The symptoms progressed to negativity, self-deprecation, and unwillingness to eat. The family had noticed weight loss of 15 kg per month (15% of baseline weight) and vomiting episodes that did not respond to symptomatic therapy. The initial clinical assessment demonstrated extracellular dehydration, a BMI of 18 kg/m^2, and functional impotence. The psychiatric assessment indicated a patient who was attentive but apathetic, remained in bed, struggled with social contacts, had emotional numbness, anger, and anxiety. She had delusions of hopelessness, punishment, and suicide ideation. The patient was hospitalized to a psychiatric department and experienced gastrointestinal discomfort, diarrhea, tachypnea, and low oxygen levels after resuming oral nutrition. Biochemical tests revealed high blood sugar, low potassium, phosphate, and magnesium levels. The patient was diagnosed with inappropriate refeeding syndrome (IRS) and was sent to an intensive care unit for 4 days. She was given parenteral nutrition and vitamin supplements (Vit B1, Vit B9, Vit D, and Vit E). The patient was subsequently readmitted to

the psychiatric facility. A diagnosis of severe depression with psychotic and melancholy symptoms was made. The patient started taking Venlafaxine at 150 mg/day in combination with Olanzapine. After 3 weeks of therapy, the patient showed improved responsiveness and reduced irritability. Despite the disappearance of delusions, functional impotence remained. The neurological exam was hard as the patient was bedridden and had flaccid tetraparesis in the lower limbs. Vitamin insufficiency was suspected to cause metabolic neuropathy. During the patient's stay in the critical care unit, their thiamine levels dropped to 36 nmol/L (normal range: 70-180 nmol/L). The patient received intravenous Vit B1 300 mg three times each day for 7 days. She was subsequently given 100 mg of vitamin B1 orally daily. Electromyography (EMG) of all four limbs revealed significant axonal polyneuropathy, mostly affecting nerves in the lower limbs (Table 1). The brain and spinal cord MRI revealed no abnormalities. The patient had acute peripheral neuropathy, most likely caused by malnutrition and melancholy depression. The patient received antidepressant medication, oral polyvitamin supplementation, and multidisciplinary follow-up (physical medicine, neurology, psychiatry).

Nerve	Latency (ms)	Amplitude (μv)	Speed (m/s)	Surface ($\mu V \times ms$)	Duration (ms)	Distance (mm)
Left median sensory						
Digit 2-wrist	3.94	9.7	35.5	15.2	3.9	140
Right superficial fibular sensory						
Leg–ankle	—	—	—	—	—	100
Left radial sensory						
Forearm-D1	3.46	3.5	28.9	1.39	2.1	100
Left sural sensory						
Leg–ankle	—	—	—	—	—	140
Right ulnar sensory						
D5-wrist	3.09	1.79	38.8	0.55	1.91	120

Table 1: Electromyography results demonstrating a considerable drop in the amplitudes of the upper limbs and an absence of the lower limbs' sensory nerve amplitude response

Discussion:

The symptoms of depression and thiamine deficiency can coexist. Thiamine deficiency first manifests as nonspecific symptoms that often appear gradually. These includes anorexia, fatigue, irritability, nausea, weakness, apathy, lack of appetite, and disturbed sleep. Food intake and appetite might change as a result of depression.[2] Nozomu Uchida et al. showed that depressed cancer patients with borderline thiamine concentrations (BTC) can quickly develop thiamine deficiency (TD). Health care providers should always be aware of the possibility of TD and the necessity of routine thiamine level testing or preventative replacement medication.[3]

Conclusion:

Recognizing and treating thiamine deficiency was crucial for persons suffering from severe depression, especially those with melancholic symptoms and considerable weight loss. Recent studies suggested that thiamine has an effect in the development of mood disorders. This indicated the importance of thiamine in preventing complications and addressing the underlying causes of mood disorders. Early identification and management can prevent long-term impairment and improve the quality of life for those with severe depression and hypovitaminosis B1.

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